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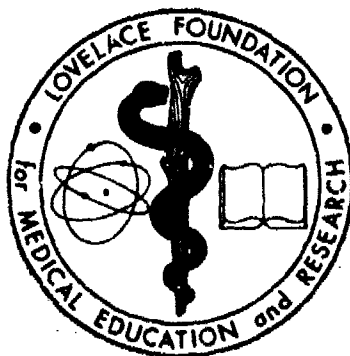
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A REVIEW OF THE TREATMENT OF UNDERWATER BLAST INJURIES

J.T. Yelverton
D.R. Richmond
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September 1976



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REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER 14 LF-54	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) A REVIEW OF THE TREATMENT OF UNDERWATER BLAST INJURIES		5. TYPE OF REPORT & PERIOD COVERED Final Technical rept. 1 Jun 74 to 30 Sep 76
7. AUTHOR(s) D. T. Yelverton, D. R. Richmond, R. K. Jones and E. R. Fletcher		6. PERFORMING ORG. REPORT NUMBER
9. PERFORMING ORGANIZATION NAME AND ADDRESS Loveiace Foundation for Medical Education and Research, Albuquerque, New Mexico 87115		8. CONTRACT OR GRANT NUMBER(s) 15 N00014-75-C-1079
11. CONTROLLING OFFICE NAME AND ADDRESS Department of the Navy Office of Naval Research Arlington, Virginia 22217		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office)		12. REPORT DATE September 1976
		13. NUMBER OF PAGES 38
		15. SECURITY CLASS (for this report) UNCLASSIFIED
		15a. DECLASSIFICATION DOWNGRADING SCHEDULE
16. DISTRIBUTION STATEMENT (of this Report) Approved for public release; distribution unlimited.		
17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)		
18. SUPPLEMENTARY NOTES This work was supported by Office of Naval Research Contract N00014-75-C-1079 with funds provided by the Naval Medical Research and Development Command.		
19. KEY WORDS (Continue on reverse side if necessary and identify by block number) Underwater-Blast Treatment Hyperbaric Chamber Underwater-Blast Injuries Extracorporeal Membrane Oxygenation Blast Pathology Positive Pressure Ventilation Air Emboli		
20. ABSTRACT (Continue on reverse side if necessary and identify by block number) Literature on underwater blast effects in man and animals was reviewed with particular reference to its pathology, pathophysiology and therapy. Anatomic structures which contain air, i.e., lungs, enteric tract, nasal sinuses and middle ear were found to be most vulnerable to blast injury. Lesions of greatest significance with respect to morbidity and mortality included air embolization, pulmonary hemorrhage and enteric perforations. The frequent association of enteric perforation in the presence of impaired cardiopulmonary function in underwater blast victims represented the most difficult medical management problem.		

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→ An historical review of therapeutic procedures used in the treatment of blast injury was then presented. Factors found to be of greatest potential benefit in improving the dismal survival rate of underwater blast victims includes: (1) prevention of air emboli, (2) maintenance of adequate ventilation and respiration and (3) timely surgical repair of enteric tract injuries. Therapeutic measures which might be used to achieve these ends included hyperbaric oxygen therapy, positive pressure ventilation and membrane oxygenation. Limitations in our current knowledge were discussed and several potentially fruitful research projects were identified.

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This work was supported by the Office of Naval Research Contract N00014-75-C-1079 with funds provided by the Naval Medical Research and Development Command. Reproduction in whole or in part is permitted for any purpose of the United States Government.

ACKNOWLEDGMENTS

Appreciation is expressed to B. Martinez and J. Miller for typing and to N. Barnett for processing the final manuscript.

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J. T. Yelverton, D. R. Richmond, R. K. Jones and E. R. Fletcher

Introduction

The mechanics and pathogenesis of blast trauma were poorly understood until World War II. During this period, acceptable interpretations of blast injury became available, (5, 24, 71, 72, 75, 87). Research efforts over the last 20 years have further clarified the knowledge gained during the war (15, 66, 67, 69, 77).

Information involving the treatment of immersion-blast victims dates back to World War I (54). The nature of the injuries and clinical signs presented by underwater blast casualties have been well-documented (8,9); but, for the most part, the therapeutic measures used in the management of these patients have not been presented in an organized fashion. This has made it difficult to evaluate the efficacy of the management procedures that have been utilized. Patients with only intra-abdominal injuries generally survived, but the prognosis was poor for casualties with chest injuries, particularly when superimposed on an acute abdomen (79).

Treatment was complicated by the fact that victims were generally not rescued until after prolonged exposure to the sea, and those with acute lung hemorrhage and air embolism did not survive long enough to receive therapy. A recent report points out that physicians are now faced with the management of these patients as a result of more rapid rescue techniques (76).

This paper will outline the lesions and clinical signs common to blast injury and present the therapeutic procedures that have been used or suggested in the treatment of immersion-blast victims.

Pathology of Blast-Induced Injury

The gross and microscopic lesions produced by air or underwater blast are primarily confined to gas containing organs and have been well documented in previous reports (10-13, 26, 30, 35, 37, 49, 71, 73, 80, 86, 87). Much of the material is based on studies on experimental animals. Inasmuch as the lesions

sustained in either air or underwater exposures are much the same, with the exception that intra-abdominal injuries tend to occur more frequently and are generally more severe in immersion blast (5, 13, 15, 24, 35, 36, 71, 73), data will be drawn from both sources for the purposes of this summary.

1. *Abdomen*

The most common intra-abdominal pathology seen at laparotomy or post-mortem examination has been to the gastroenteric tract and immediately adjacent tissues. Damage can occur anywhere from the lower part of the esophagus to the rectum, but the small and large intestine are most frequently affected. The injuries may vary from retroperitoneal, subserosal or mucosal petechiation to multiple, widespread areas of hemorrhage with laceration of the intestinal wall (10, 11, 35, 37, 86). The traumatic gut lesions found in 16 goats exposed to underwater blast, which are consistent with the findings in human cases (35, 86), have been categorized as follows (11):

- a. Isolated pin-point hemorrhages, single or in small groups.
- b. Fine linear groups of discrete or fused petechia sometimes extending in annular fashion around the lumen.
- c. Well-defined annular bands of hemorrhage, usually associated with a small amount of effused blood in the gut lumen.
- d. Broad hemorrhagic bands, produced by fusion of annular lines, with large masses of blood clot inside the lumen, attached to the mucosa of the bands. In some cases, these clots are extensive enough to completely obstruct the lumen. Perforation of the gut is common with this type of lesion.
- e. Occasional retiform markings are small and imperfectly formed or occurring as rings or extensive networks.

In general, gut hemorrhage tends to be antimesenteric and primarily mucosal. The submucosa and other coats are usually not involved except in severe cases. Free intraluminal blood is also a common finding.

Perforations of the "blowout" type (26, 86) have been reported in immersion blast victims to vary in diameter from 0.5 to 1.5 inches, and in number from 1 to 20 in any particular case (35). Hemorrhagic lesions are not necessarily associated with fresh perforations (11, 35) but zones of hemorrhage ringing

the perforations may develop later as a result of slow progressive bleeding from disrupted capillaries (35). It appears that perforations tend to occur where gas is pocketed in more or less fixed positions in the gut (30, 37, 86) and are imploded when the wave passes. If the bubbles can be displaced, only hemorrhagic lesions are likely to develop (37). Incomplete perforations are characterized by tearing of the mucosa with resulting intraluminal bleeding (10, 11, 73). Delayed perforations brought on by necrosis, secondary infection and ulceration of severely hemorrhagic sections of bowel have been described in humans (11, 34, 86) even though this possibility has been excluded by others as a result of experimental work with guinea pigs (30). Intraluminal gas distention at the sites of severe gut trauma which has been noted in monkeys may be a factor in causing delayed perforations (10).

Perforations are characterized microscopically by marked disruption of the gut wall with tearing and fragmentation of the muscle (11, 26, 86). In the small perforations, the ragged torn ends of the mucous membrane prolapse through the gap (11, 26, 71), and slight bleeding leads to the formation of a small clot that closes the wound and, in some cases, appears molded to the perforation and even projects outward from the serous coat (11) and with time may be sealed off by a thick fibrinous exudate which has been noted in histopathology sections (26, 86).

Both experimental animal results and human data have shown that other intra-abdominal organs may be affected (10-14, 30, 35, 46, 47, 66, 68, 80, 87). Hemorrhages are occasionally seen in the liver, kidneys, spleen, pancreas, adrenal glands and testes. In some cases, actual lacerations of the liver, spleen and kidneys, with resulting hemoperitoneums, have been found. The fluid-containing viscera, such as the urinary bladder and gall bladder, have generally been stated to be free of injury (10, 11, 73, 80); however, urinary bladder lesions have been reported in airblast experiments (12, 87) and in a human immersion blast victim (86).

2. *Thorax*

Pulmonary hemorrhage is the most regular intrathoracic trauma observed in either humans or other animal species. The surface lesions are generally bilaterally distributed (12, 71, 73, 80, 87) and can vary from scattered

petechiation to extensive hemorrhage of entire lobes (10, 13). The most vulnerable part of the lung is the junction between the alveolar tissue and broncho-vascular structures (12, 71). Consequently, gross lesions are generally found along the bronchi and blood vessels up to the hilus (71). Other surface hemorrhages occur in zones which are normally adjacent to firm structures, such as the heart, the vertebral column and ribs (12, 37, 71). Areas of hemorrhage are also seen in the lower lobes (12, 71, 80) at the costophrenic angle as a result of compressive pinching by the diaphragm (12). In severe cases, such as described in some immersion-blast victims (34, 35), massive hemorrhagic infiltration consolidates the pulmonary lobes and may obscure the aforementioned details (10, 12, 14). The bronchi may contain frothy and bloody fluid or clotted blood derived from intra-alveolar hemorrhage welling up into the airways (12) and from direct injury to the bronchovascular tree (10, 12, 71). Emphysematous blebs interstitial emphysema and mediastinal emphysema (10, 12, 13, 71) as well as pleural and parenchymal lacerations with resulting accumulations of blood and air in the pleural space may be evident (10, 12, 13, 37).

Histologic evidence of injury can vary from capillary rupture with slight bleeding into the interalveolar septa and separation of the epithelial layers to extensive septal rupture with bleeding into the alveoli (13, 20, 37). Hemorrhagic areas typically show large numbers of alveoli, alveolar ducts and terminal portions of bronchioles to be filled with blood (10, 12). Edema fluid may be present (12, 13, 71) but is more commonly found in sections from long-term survivors (12, 49). Damage around the bronchovascular tree, which is probably produced by differential acceleration of tissue of varying densities, is characterized by separation of the alveolar tissue from the respiratory passages and blood vessels (12, 71, 49). The perivascular and peribronchial gaps may be filled with blood, edema fluid, lymph and/or air as a result of this tissue disruption. The alveolar-venous fistulas that have been demonstrated to occur with rupture of the pulmonary veins are probably the major source of the air emboli seen in acute blast deaths (12, 49).

Injury to the bronchial wall is frequently evident even in milder forms of injury. The mucous membrane is generally the most affected (10, 12). There may be hemorrhagic sections with large areas of epithelium stripped away; elsewhere, the epithelium may be flattened and frayed in appearance with a loss of

cilia (12). In some instances, there may be considerable hemorrhage in the various layers (10,12) associated with rupture of the bronchial wall and bronchial arteries (12, 71).

Direct injury to the heart, when present, generally takes the form of superficial zones of epicardial hemorrhage (12, 71) or scattered endocardial petechiation of the ventricles, papillary muscles and occasionally along the lips of the cardiac valve leafs (12). Macroscopically visible myocardial contusions (12, 71) along with microscopically discernible muscle fiber ruptures have been noted in severely injured animals (14). Actual cardiac laceration (12), rupture (70) or hemopericardium (87) have been seen on rare occasions, whereas right ventricular dilatation is quite common (14, 87).

3. *Air Embolism*

The heart and brain can be affected indirectly by the introduction of air emboli from the disrupted lung into the coronary or cerebral circulation (5, 17, 24, 36, 71) which interrupts circulation to the myocardium or to critical portions of the central nervous system (36, 77). Air embolism has been demonstrated in several animal species (5, 12, 17, 24, 36, 66, 68, 71, 78) and human blast victims (50, 71, 76). The animal studies have shown that the incidence of air emboli is related to the extent of lung injury, is probably the most significant cause of early death, and is contributory to later cardiac failure and neurologic disorders seen in long-term survivors. Intermittent bursts of emboli related to the respiratory cycle have been detected by ultrasound in the carotid artery of a dog for a period of 30 minutes after exposure to sublethal air blast. The results of this study suggest that arterial gas emboli may be common after sublethal blast injury and are often "silent" in that there was no gross neurologic evidence of emboli during the 30 minutes following initial injury (53).

4. *Head Injuries*

Hemorrhages of the upper respiratory tract, paranasal sinuses and eardrum ruptures which are common to air blast victims (12) have been seldom mentioned in the literature on underwater blast, inasmuch as most immersion-blast victims have had their heads above water when exposed. When injury does occur, the pathological changes are similar to those in air blast (25, 37). The results

of one study with dogs demonstrate that the eardrums are susceptible to rupture if the head is submerged below the water surface during an underwater detonation, and that the severity of eardrum rupture and ossicle derangement falls off as a function of range from the explosion (69).

Diagnosis of Blast-Induced Injury

The severity of the initial symptoms and signs of immersion-blast casualties vary as a function of the distance of the subjects from the explosion and their positions in the water (5, 26, 60, 75, 79). Victims may be ambulatory at rescue and complain only of mild abdominal symptoms whereas others may be comatose and in profound shock (34). The presenting signs at admission are also greatly influenced by the time elapsed before rescue, but the gravity of the injuries are usually apparent within 6 to 12 hours after exposure (8, 79).

1. Symptoms

Survivors that are able to relate their experiences generally complain of sudden, acute abdominal pain, likened to a kick in the stomach, often accompanied by transient paralysis of the lower limbs, nausea, vomiting (with or without blood) and tenesmus. The sensation of an electric current passing through the body, testicular pain and chest discomfort followed by hemoptysis and hiccups have been mentioned (6, 8, 31, 35, 47, 60). Mild to severe shock is frequently manifested in those in which rescue was delayed or in those with severe injuries (1, 8, 34, 60). In addition, periods of unconsciousness (1, 11, 60), erratic swimming efforts (1), disorientation (41, 60), delirium (60), amnesia (41, 47) and severe residual neuropsychiatric disorder (41) have been reported.

2. Clinical Features

Patients with nonperforating intra-abdominal injuries may present with varying degrees of abdominal tenderness, guarding and some distention along with fairly normal peristaltic sounds (60). Rectal bleeding may or may not be apparent. As a rule, these individuals show a rapid improvement in status within a short period of time (35, 47, 79).

However, the persistence in abdominal signs (6) or a sudden change in condition after a period of recovery (6, 20, 34, 47) signals the development of abscesses or secondary perforations. Pyrexia may accompany the change (6) and

the blood picture may be that of a rapidly increasing leukocytosis (6, 86) with corresponding elevations in immature cells in the differential count (86).

Casualties that have sustained perforating injuries usually present with acute abdomens characterized by tenderness, guarding and board-like rigidity followed by distention, rectal bleeding and absent or diminished peristaltic sounds (60, 86). Examination by x-ray is valuable in determining the presence of free gas and fluid and complications such as obstruction and abscess (4, 8, 26, 60, 86). Sigmoidoscopy should be employed to determine the extent of low rectal injury (1, 47, 11). Frequently, the blood picture at admission is relatively normal even in cases with evident peritonitis (6, 60, 79, 86), with only a slight shift to the left in the differential count (86). Micturition may be difficult. Early confirmation of perforations is difficult, particularly if the upper abdomen is involved, inasmuch as the symptoms and signs may be referred from the chest injuries or air emboli in the abdominal vessels (24, 34). A differential diagnosis in favor of a ruptured viscus depends on the pain increasing in intensity or the persistence of an acute abdomen over a 6- to 12-hour period (8, 34, 35, 79), together with the findings from the x-ray and sigmoidoscopic examinations.

Special attention should be given to the diagnosis of the chest injuries which are commonly associated with the intra-abdominal lesions. During the early hours following blast exposure, the respiratory symptoms may be completely overshadowed by the signs of an acute abdomen (34, 35, 46, 47) that draws the attention of the examiner away from the chest (46). The extent of the lung hemorrhage is more readily estimated by radiological than by clinical methods (24, 26, 46, 47, 87) in casualties with minimal symptoms such as dyspnea and slight retrosternal pain. X-ray films frequently show various degrees of infiltration affecting from one small area to diffuse involvement of both lungs (20, 46, 47). These radiological findings increase for 24 to 48 hours and usually disappear completely within 3 to 7 days (46, 47).

Hemoptysis, tachycardia, tachypnea, cyanosis, chest pain and varying degrees of shock may also be evident at admission (46, 47). Predominate auscultatory findings will be those of localized or extensive rales and rhonci (47, 60). Bloody froth or frank blood may be seen at the nose and mouth (24, 37, 78) and moist bubbling rales are heard over both pulmonary fields in these cases

(50, 58). Chest films showing diffuse densities are indicative of pulmonary edema and hemorrhage (24, 33, 46, 60), whereas, hemothorax, pneumothorax, pneumomediastinum or interstitial emphysema are apparent if the lungs are lacerated (46, 76).

Streaming bubbles or pale silvery sections in the retinal vessels, retinal pallor, or sharply defined areas of pallor on the tongue, may indicate the presence of air emboli (24, 76, 78).

Extensive damage to the pulmonary parenchyma causes aberrations in the ventilation perfusion ratio, lung diffusion capacity and increased venous-arterial shunting (23) with resulting hypoxemia reflected by a reduction in the arterial oxygen tension (47). Hemodynamic disturbances of the pulmonary circulation are evidenced by a reduction in arterial blood pressure and elevation in central venous pressure which can lead to acute right heart failure (15, 18). Chest films showing cardiac enlargement with some straightening of the pulmonary concavity and widening of the azygos vein may be signs of such failure (46, 47). Electrocardiograms demonstrating changes compatible with cardiac injury complete the clinical picture (24, 47, 50).

Since respiratory failure may develop as part of the syndrome of post-traumatic pulmonary insufficiency (56, 58, 76), all immersion-blast victims, particularly if multiple injuries are present, should be closely observed for signs of increasing respiratory distress and hypoxia resulting from a buildup of edema fluid, continued lung hemorrhage and atelectasis. When possible, pulmonary function studies, serial arterial gas measurements, determination of the hourly urine output and serial x-ray examinations of the chest are particularly important in these patients (58).

Treatment of Blast-Induced Injury

The management approach for immersion-blast victims is similar to that for patients suspected of having total body trauma (85). Any person that has been in the vicinity of an underwater blast should be admitted for observation for a minimum of 48 hours (76) or longer if abdominal symptoms are present (47, 79). There has been one reported case of post-traumatic pulmonary insufficiency developing 48 hours after exposure in an initially asymptomatic patient (76). Other cases presenting with acute abdomen may demonstrate a rapid improvement only to suffer from delayed perforations a week later (47, 79).

The prognosis has generally been grave for casualties with obvious lung injury (8, 34, 79), but if they survive the first 24 hours, clinical conditions frequently clear rapidly with convalescence as early as 3 weeks (79). The most difficult treatment problems arise in those individuals with serious lung and intra-abdominal injuries which may be complicated by arterial air embolism (76). Survival of these casualties depends on rapid rescue and immediate resuscitation.

Individuals with multiple traumatic lesions may respond to initial resuscitation only to expire from cardiorespiratory failure (47) resulting from the development of progressive pulmonary insufficiency or cardiac insufficiency during surgery or some hours to days later. Progressive pulmonary insufficiency, or the so-called shock lung syndrome, can be precipitated by direct-blast injury to the chest, bleeding into the upper airway, aspiration of gastric contents, abdominal distention, retention of bronchial secretions, sepsis or shock incident to hypovolemia and serious wounds (56, 58). Iatrogenic factors such as misuse of blood, crystalloids, vasopressors, anesthetics, sedatives, oxygen, respirators (employing large tidal volumes and high airway pressures) and immobility aggravate the condition (7, 56, 58).

Although opinions in the literature vary with regard to the initial treatment of immersion-blast victims, the general approach is theoretically quite simple: complete bed rest; sedatives and antitussives for restlessness and coughing, respectively; avoidance of food or fluid by mouth until extent of injury is determined; adequate airway maintenance; humidified oxygen administration; relief of air embolic symptoms; assurance of adequate lung expansion and avoidance of fluid therapy when chest injury is apparent except in extreme cases.

1. *General Medical Management*

A prone (36) or reclining position, with the head low in a left-lateral-prone orientation (78) or Trendelenberg position (73) has been suggested for cases with severe lung involvement to promote airway drainage and to prevent migration of air emboli to the heart and brain during the early hours following injury. Individuals that are being maintained with their heads lowered should be closely observed for signs of venous distension (78). After the air embolic danger has passed, either a semi-sitting (79) or a high Fowler's (6, 86) position will ease the work of breathing. In any event, a single posture should not

be maintained for extended periods of time, as a fixed position may encourage fluid accumulation and atelectasis. If the patient cannot contribute to his own physiotherapy, periodic full turning from side-to-side is essential (7, 42, 56).

The cautious use of sedatives and analgesics is necessary to relieve pain and restlessness if complete rest is to be achieved (11, 24, 78-80). Intravenous administration of these drugs results in immediate pain alleviation and permits the use of repeated small doses thereby precluding the threat of respiratory depression (58). Meperidine, 10 to 25 mg; morphine sulfate, 2.5 to 5.0 mg (56, 58); ethylmorphine hydrochloride, 30.0 to 100.0 mg; and dihydromorphine hydrochloride, 2.0 to 4.0 mg with atropine sulfate, 0.25 mg (24), seem to be quite satisfactory. Scopolamine-eukodal-ephedrine has produced good results in small doses (24) and diacetylmorphine in 8-mg doses has also been used effectively (79, 80). Slow injection of 30 to 40 cc of 30 percent alcohol has been recommended for its euphoric and slight anesthetic effects (24, 78). Sedation with morphine in conjunction with muscle relaxants cuts down O_2 uptake by as much as 25 percent thereby relieving some of the O_2 demand of hypoxic patients (43).

Appropriate antibiotic therapy should be instituted, when indicated, to prevent and control infection (47, 76).

2. *Management of Intra-Abdominal Injuries*

Fluids and food should not be given by mouth until the extent of injury is determined (73, 79). Continuous gastric and intestinal aspiration may be necessary to relieve distention (60, 73, 79, 86) incident to paralytic ileus as well as to reduce the danger of delayed intestinal perforation and vomiting episodes. The reduction of intraluminal pressure relieves the tension on severely traumatized sections of intestine (73), and also relieves pressure against the diaphragm thereby allowing better ventilation of the lungs (56, 58). Intestinal decompression should be continued until effective peristalsis returns.

Patients with nonperforating intra-abdominal injuries respond well to conservative treatment demonstrating rapid relief of symptoms within 24 to 72 hours (34, 35, 79, 80). However, these individuals should be observed for 7 to 10 days for signs of delayed perforation, particularly if bloody stools have been evident (47, 79).

The treatment of cases with perforating lesions, in the absence of pulmonary injuries, does not differ fundamentally from that of other nonpenetrating abdominal injuries (35). Small bowel injuries are repaired by primary suture (34, 35, 47) and injuries to the colon are usually treated by exteriorization of the lacerated segment or suture repair, with a proximal colostomy (34, 47). One therapeutic regime which has been effectively employed for small perforations and peritoneal inflammation involves the use of morphine, indwelling intestinal suction drainage and intravenous infusions of electrolyte, dextrose and plasma (1).

Major complications arise when chest involvement is superimposed on an acute abdomen (8, 34). These patients have frequently been denied surgery as a result of a compromised cardiopulmonary status only to succumb to peritonitis and those that have received surgical treatment often expire during or shortly after laparotomy (6, 62, 47). It has been suggested that surgery be delayed for 24 to 48 hours after injury to allow stabilization of the cardiopulmonary circulation (24, 40, 79), but others have felt that early laparotomy is essential in view of the high incidence of colonic perforations (47).

3. *Management of Intrathoracic Injuries*

An adequate airway must be maintained (20, 47, 58, 76, 86) and mechanical aspiration by nasotracheal suction, bronchoscopy or tracheostomy should be carried out when indicated (47, 58). Detergents in mists and alcohol vapor have been suggested to facilitate airway clearance (78), whereas, the increased airway resistance associated with primary blast injury (16) might be reduced by the mist application of bronchodilator, such as Isoproterenol. Coughing, which is advocated for clearing secretional obstruction of the lower airways (58) should be held to a minimum for 7 to 10 days to avoid dislodging of blood clots and reopening any communication between the airways and the pulmonary vascular system (78). Once the patient has stabilized, mild breathing exercise should be instituted (24).

Airway maintenance through the use of endotracheal intubation or tracheostomy tube is necessary if mechanical ventilation is indicated for prolonged periods (56, 58) and if gastric contents have been aspirated, endotracheal intubation should be performed immediately to remove the gastric contents (58).

Adequate concentrations of humidified oxygen should be provided to correct hypoxia (20, 35, 47, 60, 73, 79, 86) and relieve minor air embolic symptoms by lowering the partial pressure of nitrogen in the lungs (17, 28, 36). However, oxygen therapy to correct hypoxia should be employed with caution, inasmuch as it may be a factor in maintaining or reinitiating hemorrhage (17, 59, 78) as well as producing toxic side effects in the blast-injured lung. The use of helium-oxygen mixtures (78) might prove effective in this regard, by providing for adequate arterial and tissue oxygenation with a minimum acceptable increment in inspired oxygen tension ($P_{I_{O_2}}$) while furnishing a zero gradient for nitrogen elimination. An arterial oxygen tension ($P_{a_{O_2}}$) range of 70 to 100 mg Hg may be considered acceptable for previously healthy individuals with shock lung if their cardiac output is adequate (42, 56). A 30-minute test inhalation of 100 percent oxygen will define the lowest $P_{I_{O_2}}$ compatible with an acceptable $P_{a_{O_2}}$ (52). Patients with severe lung injury will probably fail to show a significant improvement in the $P_{a_{O_2}}$ on 100 percent oxygen, which is indicative of a large venous-arterial shunt. Venous-arterial shunts exceeding 30 percent of the cardiac output have been reported for dogs and sheep with extensive blast-lung hemorrhage (23), resulting in a condition in which the $P_{a_{O_2}}$ is below the level required for full hemoglobin saturation with the animal spontaneously breathing pure oxygen at atmospheric pressure (15, 23).

On a short-term basis, severely hypoxic individuals refractory to 100 percent oxygen at ambient pressure and with definite evidence of arterial air embolism would benefit from hyperbaric oxygen. This mode of therapy has been promoted for treatment of blast-introduced air embolism (5, 17, 36), and has been used to eliminate air emboli introduced during cardiac surgery (48, 83). It has been suggested that blast-injured victims with signs of respiratory distress might fare best if secondary and tertiary injuries are treated first, an adequate airway ensured and then early compression to 2.0 ata with 100 percent oxygen instituted (76).

Compression to 4 ata with a 90-minute decompression has been utilized to relieve air embolic symptoms and increase survival in blast-injured rabbits (17). Patients affected by surgically induced massive air embolism have been effectively treated by compression to 6 ata on air for 2 hours followed by a 36-hour decompression in accordance with U. S. Navy Treatment Table 4. Oxygen was

not utilized until pressures below 2.5 ata were reached to circumvent the toxic effects of oxygen on the lung and brain at high pressures (74, 83). A more brief treatment schedule utilizing a helium-oxygen (80%-20%) mixture, administered via a Bird Mark VII respirator, at 6 ata for 9 minutes followed by 100 percent oxygen at 2.8 ata for a total chamber stay of 5 hours 13 minutes has also produced good results (48). Results of a recent study demonstrate an increase in survival time and recovery for blast-injured guinea pigs, rabbits and dogs receiving hyperbaric oxygen (22). A 29-hour schedule at an initial $P_{I_{O_2}}$ of 1.5 ata was beneficial for rabbits; whereas, a beginning $P_{I_{O_2}}$ of 2.0 ata was required for guinea pigs. Dogs proved even more resistant in requiring a 24-hour schedule with 100 percent oxygen starting at 2.2 ata followed by an additional 24-hour treatment with 60 percent oxygen at 1.0 ata.

Regarding oxygen toxicity, the prolonged use of a high $P_{I_{O_2}}$ can induce severe pulmonary damage in normal lungs (42, 84). Abnormal lung pathology developed after 40 hours of continuous ventilation on 100 percent oxygen at 1 atm in patients with irreversible brain damage; whereas, there was no evidence of pulmonary oxygen toxicity with a mean exposure of 24 hours to 100 percent oxygen at 1 atm in individuals recovering from open heart surgery (42). Surfactant measurements and autofluorescence of alveolar lining membranes of rats or guinea pigs breathing varying concentrations of oxygen at 1 atm remained normal during the first 48 hours of exposure and became abnormal just before death at 55 to 60 hours (84), and a sharp reduction in surfactant has been seen in dog lungs after 48 hours of exposure to pure oxygen (57). However, oxygen toxicity is apparently not only governed by alveolar oxygen pressure ($P_{A_{O_2}}$) but also by $P_{a_{O_2}}$ (82). The large alveolar-arterial oxygen gradient $(A-a)_{O_2}$ resulting from continued perfusion of blast-injured, nonventilated regions of the lungs (23) might render such cases more resistant to oxygen toxicity (22), and some studies have indicated that animals with lung injuries are less affected by a high $P_{I_{O_2}}$ than normal animals (59, 82). Thus, one might consider the administration of 100 percent oxygen at 1.0 ata for periods of time ranging up to 48 hours. It is probable that a 60 percent oxygen mixture at 1.0 ata can be tolerated for indefinite periods of time (7).

Intermittent positive pressure ventilation (IPPV) or continuous positive pressure ventilation (CPPV) with variable oxygen concentrations to restore

an adequate Pa_{O_2} , to reduce the work of breathing and to assure adequate lung expansion are the treatments of choice in shock lung (55, 76). However, any therapy involving intrapulmonary application of inspiratory positive pressure has previously been felt to be contraindicated for blast victims (17, 36, 78). It was suspected that full expansion of the lungs would increase pulmonary hemorrhage, force air into the pulmonary veins (17, 36, 40, 59) and could cause the development of a tension pneumothorax if alveoli had ruptured into the pleural space (21, 47). The results of one study suggest that the time of administration of IPPV may be an important factor in determining the success of treatment (21). Critically injured dogs that received IPPV with pure oxygen immediately following blast exposure did not respond to treatment; whereas, animals that were maintained on 100 percent oxygen at 1.2 atm for 4 hours in a hyperbaric chamber followed by 2 hours of ventilation with 100 percent oxygen survived until taken off the respirator. Both immersion blast and airblast victims with severe respiratory distress have been treated with positive pressure and oxygen with varying degrees of success in spite of theoretical objections (47, 55, 76). Regardless of the type of respirator used, the machine should be set to deliver gas at the lowest positive pressure feasible to maintain the O_2 and CO_2 arterial tensions within the normal range in order to avoid over distention of the lungs (21). The guidelines which have been suggested for the commencement of mechanical ventilation in nonthoracic respiratory failure (61) might be considered for immersion-blast victims:

Indications for Respiratory Support

		Acceptable Range	Chest Physical Therapy, Oxygen, Close Monitoring	Intubation Tracheotomy Ventilation
Mechanics	Respiratory rate	12-25	25-35	> 35
	Vital capacity, ml./kg.	70-30	30-15	< 15
	Inspiratory force, cm. H ₂ O	100-50	50-25	< 25
Oxygenation	A - aDO ₂ , mm. Hg ^a	50-200	200-350	> 350
	P _a O ₂ , mm. Hg	100-75 (air)	200-70 (on mask O ₂)	< 70 (on mask O ₂)
Ventilation	V _D /V _T	0.3-0.4	0.4-0.6	> 0.6
	P _a CO ₂ , mm. Hg	35-45	45-60	> 60 ^b

^aAfter 15 minutes of 100% O₂

^bExcept in chronic hypercapnia

(61) Pontoppidan

Extracorporeal membrane oxygenation may be considered for a severely hypoxic victim whose condition is refractory to the aforementioned modes of therapy or for one whose condition is such that prolonged maximum respiratory support is indicated. Clinical studies have demonstrated that long-term extracorporeal oxygenation for periods of time up to 17 days is feasible in patients with non-blast induced acute respiratory failure and refractory hypoxemia (2, 3, 27, 45, 51, 64). One moribund immersion-blast victim has been maintained on bypass for 60 hours (76). The low survival rate achieved with this procedure is largely due to the fact that extracorporeal circulation (ECC) has generally been instituted beyond the point where any survival by conventional methods can be expected (3, 44) and after iatrogenic lung damage from prolonged use of 100 percent oxygen and ventilators has occurred (2, 3, 44, 45). Individuals with direct pulmonary capillary or alveolar trauma in which the principal functional alteration is increased venous admixture have the best chance of recovery with ECC if sepsis can be controlled (3, 51).

Either venovenous or venoarterial circuits are generally utilized for ECC. The venovenous cannulation system does not produce any significant detrimental hemodynamic changes and avoids the dangers of arterial dissection, embolization (51), thromboembolic infarction and pulmonary liquifaction necrosis (63) associated with venoarterial circulation. However, near-total bypass can be achieved with the venoarterial circuit thereby allowing a higher step-up in P_{aO_2} , a reduction in pulmonary artery pressure and right ventricular work load, a possible decreased transudation of fluid into the interstitial tissue of the lungs, and a greater reduction in FI_{O_2} and high respiratory pressures (44, 51). Recent findings indicate that the potential deleterious effects of venoarterial perfusion may be negated if partly venous and partly arterial returns are used (63). Two problems associated with either bypass technique have been the regulation of heparin requirements and platelet loss to the extracorporeal components (29, 45). Experimental work with extracorporeal components ionically bound with tridodecylmethylammonium chloride and methyltricaprylammonium chloride heparin complexes suggest that systemic or regional heparinization may not be necessary (38, 65). There are also some data available indicating that precoating of bypass components with albumin (32), and the elimination of blood-gas interfaces (52) will reduce platelet loss.

Until the risk involved with the use of ECC becomes so small that its application will pass into the category of prophylactic therapy, one should probably consider the following guidelines (2, 27, 44, 76) prior to instituting ECC in patients with severe blast-lung injury: (1) The P_{aO_2} should be consistently below 40 to 50 mmHg with the patient on 100 percent oxygen and receiving maximal respirator support. Since early use of positive pressure ventilation with high pressure appears to be contraindicated, 50 mmHg should probably be considered the cut-off point. (2) The pulmonary lesion is reversible, preferably within a period of 10 to 15 days. (3) There is evidence of hypoxemic myocardial and/or CNS depression. (4) Major bleeding (nasopharyngeal, tracheal or intra-abdominal) is not present to contraindicate the use of anticoagulants. (5) There is convincing evidence that the patient is progressing to a moribund state.

The control of further pulmonary bleeding, for which intravenous injection of alcohol has been recommended (24) and the prevention or reduction of

pulmonary edema with diuretics (47, 76) should be an integral part of respiratory care. If diuresis is established and fluids restricted as soon as possible in cases with severe respiratory insufficiency, excessive edema may be avoided (7). Hemodialysis may be necessary if renal function is insufficient (19).

Steroid administration which has not been utilized until recently (47, 55, 76) should be considered for use both as a prophylactic and a therapeutic agent. Pharmacological doses, 15 to 30 mg/kg, of methylprednisolone sodium succinate (Solu-Medrol[®]) have proven to be effective in preventing much of the cellular damage and tissue destruction associated with a shock-lung state (81). Intravenous doses of frusemide and hydrocortisone have produced good results in treating interstitial pulmonary edema incident to airblast injury (55).

4. *Management of Cardiovascular Complications*

Congestive heart failure may be a significant management problem if complete rest and respiratory therapy fail to relieve the hypoxia and pulmonary hypertension associated with severe lung hemorrhage. Instances of fulminating cardiac insufficiency have been described as long as 5 days after injury (50).

Acute cardiac failure may be treated by rotating tourniquets, venesection and rapid digitilization (39, 40, 50). Stropanthin has been suggested for its extra-rapid action (24). Caffeine initially administered intravenously in large doses as a cardiac stimulant and diuretic and later by mouth in the form of coffee may be beneficial providing that it does not interfere with sleep (24). The application of mild heat would also be helpful (24, 79).

Intravenous fluids, plasma or blood should not be used to correct hypotension unless absolutely necessary (34, 35, 39, 40, 78, 79, 80). Unless solid organ rupture is definite, the finding of lowered blood pressure is probably not due to blood loss but can be explained by air emboli, vagal reflex or pulmonary hypertension (5, 46). Overloading may increase the existing pulmonary insufficiency and may throw the already compromised right heart into congestive failure (79, 80). Where intraperitoneal fluid losses are to be expected in cases with combined abdominal and pulmonary lesions, arterial and central venous pressure, blood volume, plasma osmolarity and urinary output measurements should be used to guide fluid replacement (47).

5. *Anesthetic*

Numerous authors have noted the poor tolerance of patients with serious lung injury requiring early surgery to general anesthesia (24, 35, 47, 50, 73, 80). However, the choice of other methods is limited. Local anesthesia would probably not provide a sufficient surgical field (35, 80). Spinal anesthesia may be considered as an alternative (47), but it is contraindicated in patients with any degree of shock or reduced blood pressure (35, 80). Cyclopropane has been suggested as the ideal anesthetic in these cases, inasmuch as it is given with a high percentage of oxygen, requires little reinforcement to secure good muscle relaxation and has little depressant effect (35). Nitrous oxide and oxygen preceded by a minimum dose of pentothal or evipan has also been recommended (80).

Conclusions and Recommendations

Recent advances in rescue techniques and medical technology have made it feasible to save moribund patients with multiple primary-blast injuries. The probable existence of some degree of air embolism in these individuals suggests initial resuscitation with hyperbaric oxygen, after establishing an adequate airway, would eliminate the air embolic symptoms and improve tissue oxygenation. Recent studies indicate the effects of hyperbaric treatment in blast injury depend on the duration of treatment and the PO_2 of the inspired gas and to some extent, are independent of chamber pressure. This suggests the use of portable low pressure hyperbaric units in rescue operations. Individuals with extensive air embolic occlusion would probably require additional compression to 6 ata to enhance the rate of resolution of symptoms by reducing bubble size which would improve circulation to affected parts of the heart and brain. This could be accomplished after transfer to more permanent hyperbaric facilities. After the air embolic danger has passed, some form of positive pressure ventilation should probably be instituted to restore an adequate arterial oxygen tension, reduce the work of breathing and assure adequate lung expansion. The use of helium-oxygen mixtures would eliminate oxygen toxicity problems as well as reduce respiratory effort. The use of high ventilatory pressures which would probably be required for severely injured patients could be circumvented by extracorporeal membrane oxygenation.

Future studies should be planned to: (1) determine the time period when positive pressure ventilation can be instituted without the danger of reintroducing air emboli; (2) determine the effects of long-term extracorporeal bypass on blast-injured subjects; (3) determine the best anesthetic regime to be used in performing laparotomies on patients with lung injuries superimposed on acute abdomens and (4) determine if prophylactic therapy can postpone the necessity of early surgical intervention in subjects with acute abdominal lesions.

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